OTHER DEVELOPMENTS

AUSTRALIA

[44] Drivers Told to Throw Away Car Cigarette Lighters

The Queensland Cancer Fund is reportedly encouraging drivers to throw away their cigarette lighters and install a flashlight in its place as part of its efforts to raise awareness of the "dangers of passive smoking in cars." *See Northern Times*, December 10, 1993.

AIRLINES

[45] World Airline News

- Asia
  On December 11, 1993, Asiana Airlines announced it will ban smoking on flights of up to six hours. The ban, due to go into effect on January 1, 1994, will affect flights between Seoul and Singapore as well as flights from Seoul to Bangkok, Saigon, Saipan and Tianjin. *See Korea Economic Daily*, December 13, 1993.

- China
  China Airlines is said to be adding two flights each week between Taipei and San Francisco. The new flights will reportedly be nonsmoking. *See Aviatian Europe*, December 23, 1993.

- Korea
  Korean Air reportedly announced it will ban smoking on flights to Canada. Although no effective date has been scheduled, the airline says it plans to phase in the ban on its Seoul-Vancouver-Toronto route beginning in 1994. *See Korea Economic Daily*, December 20, 1993.

- New Zealand
  A newswire service reports that an Air New Zealand passenger spent a day in jail and was fined $A90 after lighting a cigarette during a nonsmoking flight to the United States. He was reportedly incarcerated and fined by the Cook Islands High Court. *See Australian Associated Press*, January 1, 1994.

- Singapore
  Effective December 1, 1993, Singapore Airlines reportedly announced a smoking ban on its flights between Singapore and Australia and has introduced nonsmoking flights between Australia and the United Kingdom. A press report indicates the airline had been offering some smoke-free flights to and from London since September 1. The airline claims to offer 560 nonsmoking flights of its total 840 weekly. *See Aviation Daily*, December 2, 1993.

- Thailand
  THAI Airways will reportedly begin introducing nonsmoking flights on February 15, 1994. According to a press report, affected routes will include Bangkok to such cities as Hong Kong, Singapore, Malaysia, Burma and its flights to Indochina. *See Bangkok Post*, December 23, 1993.

- United Kingdom
  On January 1, 1994, nonsmoking flights lasting up to 24 hours, will go into affect during a trial period for British Airways' flights between Australia and New Zealand. The airline says the flights will become permanently nonsmoking if passenger feedback is positive. *See Press Association Newsfile*, December 31, 1993.

MEDIA COVERAGE

[46] "Children Breathe Easier with Controls on Factory Fumes," *South China Morning Post*, December 17, 1993

A University of Hong Kong study claims that breathing problems among children have improved since controls were put on factory chimney fumes but that the gains were negated for children of families who smoke. While the study estimates that 18,000 visits to clinics were averted by having cleaner air, it reports that children exposed to tobacco smoke in the home continue to be at "much higher risk" for breathing problems. This is reportedly the first time such data on ETS have been reported in Hong Kong.

APPENDIX A

The numbers assigned to the following article summaries correspond with the numbers assigned to the synopses of the articles in the text of this Report.

LUNG CANCER

[27] **"Dietary and Mental Health Differences Between Never-Smokers Living in Smoking and Non-Smoking Households," D.H. Thompson and D.M. Warburton, *Journal of Smoking-Related Disorders* 4(3): 203-211, 1993**

"In this paper, we present some evidence of the confounding effect of diet on studies of the association of ETS with an increased risk of cancer and heart disease."

"In view of this fragmentary evidence that studies of the effects of ETS may be confounded by differences in lifestyle between ETS-exposed and non-smokers, who are not exposed to ETS, we investigated these differences with particular reference to diet, alcohol consumption and mental health of a large sample of never-smokers living in smoking and non-smoking households."

"Our results show important lifestyle differences between never-smokers living in smoking households (SH) and never-smokers living in non-smoking households (NSH). SH consume fats more frequently, drink more alcohol and eat fewer root vegetables and cereal. In addition, SH have poorer mental health than NSH. We now consider some of the health implications of these differences."

"Our results show that SH are more likely to consume chips, whole milk, butter, crisps, sausages and are more likely to fry their food than NSH. As a consequence, this group will be consuming a greater quantity of saturated fats."

"Our results show that SH are less likely to consume root vegetables (including carrots) and fresh fruit juice than NSH."

"The above results show a possible confounding effect of dietary carotene intake when studying the risk of developing lung cancer. Therefore, it is important to take into account studies which have shown a strong relationship between lung cancer risk and carotene intake. Whether dietary differences are sufficient to explain completely the reported increased relative risk of lung cancer in those exposed to tobacco smoke is unknown. However, it is true that nearly every study of ETS and disease has failed to take such dietary differences into account."

"If the data for alcohol consumption in the previous week are representative of typical consumption, there are a greater number of non-drinkers, heavier drinkers and fewer moderate drinkers in SH."

"Our results show that a greater proportion of SH never drink coffee while a slightly greater percentage drink over five cups per day. It is difficult to establish the health consequences for such differences in consumption."

"Our results show that SH were more likely to be experiencing insomnia or to report higher rates of depression than NSH. This conclusion is based on replies to the four questions: 'Face up to your problems?', 'Everything getting on top of you?', 'Unhappy and depressed?' and 'Losing confidence in yourself?'. In addition more of those in NSH had been 'Keeping busy and occupied?'"

"It is not possible to establish whether the respondents in the present study would have met diagnostic criteria for major depression. However, it seems clear that an individual's state of mind can have an important effect on physical well-being and so represents a negative health indicator for SH."

"There are a number of differences between never-smokers living in smoking and NSH which have adverse implications for health and are confounding factors in the study of the health consequences of ETS. Future studies of ETS and health should control for these factors. Also, from the database we are unable to determine the diet of children of the respondents."

"However, it would seem a reasonable assumption that children living in SH will have similar diets to the adults in the household and so a less healthy diet than children living in NSH. Given the association of poor diet and proneness to infection, it might be expected that there would be a higher incidence of infection among children living in SH."

Cardiovascular Issues

[28] **"Indoor Passive Smoking and Cardiac Performance: Mechanisms Able to Cause Heart Failure," L. Mori, F. Bertanelli, P. Fabiano, A. Battaglia, and A. Leone, *Journal of Smoking-Related Disorders* 4(3): 213-217, 1993**

"Nineteen non-smoking male volunteers -- nine healthy and 10 with a previous myocardial infarction, underwent exercise stress testing twice: once in a smoke-free environment and once in a smoking environment (carbon monoxide concentration 30-35 ppm)."

"In healthy people, we observed the following change after exercise in a smoking environment: mean prolonged time to recovery to pre-exercise heart rate [19 min vs. 8.5 min]. Survivors of infarction showed a significant reduction of the peak of exercise [80 watts vs. 120 watts], prolonged time to recovery to pre-exercise heart rate [21 min vs. 12.3 min], increased blood carbon monoxide concentration [2.3% vs. 1.2] and cardiac arrhythmias."

"The results obtained seemed to depend on environmental hypoxia due to carbon monoxide for healthy people and environmental hypoxia associated with increased post-exercise blood carbon monoxide concentration for survivors of infarction."

"Two main questions arise from the results of this study. First, the role of passive smoking as a factor for progressive cardiac damage, which is influenced by the degree and type of exposure to smoking. Second, the different pathological mechanism able to impair cardia function in healthy and diseased subjects."

"Heart disease has emerged as an important consequence of environmental tobacco smoke (ETS). There is epidemiological evidence that passive smoking may cause approximately a 30% increase in the risk of death due to heart disease. Moreover, ETS reduces exercise ability in ischaemic patients by up to 30%.

"Either acute or chronic exposure to passive smoking may damage the cardiovascular system. Chronic exposure may lead to ischaemic heart disease, whereas acute exposure impairs cardiac function, as our studies show. Moreover, cardiac impairment begins to be quite evident at low blood and environmental CO concentrations in survivors of acute myocardial infarction. This latter effect seems to depend on three factors: the environmental CO concentration, blood CO concentration and individual health."

"To our knowledge, no study closely links passive smoking and acute heart failure....At present, the link between passive smoking and heart failure remains an area of doubt and intensive debate."

"Perhaps future studies should assess the effect of passive smoking on various aspects of cardiac pathology in patients who already suffer from some form of ischaemic heart disease."

Respiratory Diseases and Conditions -- Children

[29] **"Effects of Passive Smoking on Respiratory Illness from Birth to Age Eighteen Months, in Shanghai, People's Republic of China," C. Jin and A.M. Rossignol, *Journal of Pediatrics* 123: 553-558, 1993**

"The objective of this study was to evaluate the effects of household environmental tobacco smoke, in the absence of maternal smoking during pregnancy, on the cumulative incidence ('risk') of hospitalization for respiratory illness in infants from birth to age 18 months, with the use of data from a census of children residing in Shanghai, People's Republic of China."

"This study identified a significant dose-response relationship between daily household cigarette use and the cumulative incidence of respiratory illness in children from birth through age 18 months. This relation could not be explained by in utero effects of maternal smoking nor by any of the potential confounding factors evaluated. Being fed human milk for at least 1 month was identified as a preventive factor for respiratory illness and was independent of the effects of household cigarette use or number of household members. The latter variable, however, was relatively constant among the households studied. The effect of the failure to breast-feed an infant was estimated to be approximately equal to the effect of exposing an infant to household use of 10 to 19 cigarettes per day."

"The present study showed a gender difference in the 18-month cumulative of respiratory illness: boys appeared to be more affected than were girls."

"In Shanghai, the indoor air quality for households using coal stoves has been reported to be worse than the air quality for households using gas stoves. In our study, however, children living in households using coal stoves had a lower cumulative incidence of respiratory disease than did children from households using gas stoves. One possible contributing explanation for this result is that children spend most of their time in the parents' bedroom, where the fathers smoke, where as the cooking stove is located in the kitchen."

"Two potential sources of error in this study warrant discussion. The first is that exposure to passive smoke is not an easily quantifiable variable. Most studies of long-term health consequences of passive smoking have relied on the smoking status of parents or their household members as the basis for defining exposure status. In our study, the exact number of cigarettes smoked at home by each family member was difficult to ascertain accurately....Most of the smokers in this study were the children's fathers, who left their homes for their places of employment during workdays; the remainder were grandfathers and uncles who lived in the same household."

"The second potential source of error is recall bias....In our study, however, this type of recall bias did not appear to be a substantial problem, because there was little difference in the number of episodes of respiratory illness reported per person by different respondents."

"This study demonstrated significant negative effects of household smoking on the respiratory health of Shanghai infants. Since the data were collected in 1985, the economy and lifestyle in China have changed substantially because of the 'open door' policy. Smoking in young women is becoming fashionable, but the prevalence of smoking in male adults is stable at more than 70%. Under these circumstances, the public health effects of passive smoke on children's respiratory health documented in this study probably underestimate the current effects."

OTHER CANCER

[30] "Environmental Tobacco Smoke and the Risk of Cancer in Adults," J. Tredaniel, P. Boffetta, R. Saracci, and A. Hirsch, *European Journal of Cancer* 29A(14): 2058-2068, 1993

"This paper will review the epidemiological evidence between ETS exposure and cancer in adults, with special emphasis on cancers in organs other than the lungs."

"It is very unlikely that chance alone might explain the association between ETS and lung cancer: even if the increase in risk is not likely to be higher than 40%, the size of the populations which have been studied is large enough to exclude with reasonable confidence the possibility that it originated only by random statistical variation; overall, 3453 lifelong non-smoking lung cancer cases have been included in the published studies, 2716 of them (79%) after the 1986 reports which already concluded on the carcinogenic role of ETS. On the other hand, considering that the association between ETS exposure and cancers in adults other than lung cancer, if existing, is presumably weak, one must wonder whether chance alone could explain the reported findings. In addition, most of the studies which we have reviewed, especially the initial ones, although sometimes suggestive of a positive effect, have had several deficiencies which can substantially bias study results, increasing the difficulty in their interpretation, especially if the likely effect of ETS is small. Finally, unrecognised confounding factors may have produced spuriously positive results."

"The criteria to consider, in judging whether an association observed between a particular factor and disease is one of cause and effect, have been extensively discussed. All the available data seem to fulfill, at the present time, and at least to reasonable degree, the criteria needed to accept a causal link between ETS and lung cancer among lifelong non-smokers."

"The problem is much more complicated when dealing with ETS and cancers other than the lung. A dose-response relationship has not been considered in the majority of the studies. Few studies attempted to characterize exposure more specifically than using the numbers of smokers at home."

"Biological plausibility must especially be questioned."

"In particular, the results of elevated risk of sinonasal cancer -- in addition to the risk of lung cancer -- strengthen the plausibility of carcinogenic hazards of sidestream smoke inhalation through the nose."

"When found, the association between ETS exposure and cancers not related to active smoking is difficult to interpret, and necessarily regarded with caution. We may be seeing the effect of unrecognised confounder(s). On the other hand, other possible mechanisms may be involved."

"The ubiquitous presence of tobacco smoke in homes, workplaces, public and private areas has made, until recently, exposure to ETS virtually unavoidable. Involuntary exposure to tobacco smoke has only been intensively investigated as a risk factor for disease in non-smokers in the past decade. Consequently, the evidence on ETS is more limited in scope than for active smoking, and controversy remains concerning the association of ETS with certain diseases. Although ETS-related lung carcinogenesis can be considered as definitely established, there is, as yet, no final evidence of an association between ETS exposure and cancer at sites other than the lung. However, such a conclusion is still based on limited information, and considering the large number of studies which have resulted in widely divergent findings, methodologically improved studies with larger sample sizes are needed. There are sites such as the nasal cavity and the sinuses for which the available evidence strongly suggests the presence of an effect, while for other sites, such as the urinary bladder, it rather suggests the absence of an association. The suggestion of an effect on other sites, such as the uterine cervix, the brain and the breast is more difficult to interpret. Yet, full resolution would seem unnecessary for the evolution of public policy on ETS, an air pollutant with a readily controllable source. Our priority must be to continually encourage the reduction in tobacco use.

Other Health Issues

[31] "Day Care Attendance and Other Risk Factors for Invasive *Haemophilus influenzae* Type b Disease," C. Arnold, S. Makintube, and G.R. Istre, *American Journal of Epidemiology* 138(5): 333-340, 1993

"The introduction of an effective vaccine makes implementation of a vaccination program against *Haemophilus influenzae* type b (Hib) disease a major public health concern."

"We conducted a population-based matched case-control study of all reported cases of invasive Hib disease occurring in the state of Oklahoma in 1986 and 1987. We examined the role of previously reported risk factors: day care, young siblings in the home, crowding in the home, tobacco smoke pollution, socioeconomic status, race, breast feeding, and Hib polysaccharide vaccine."

"Our primary analysis focused on the risk of Hib disease associated with day care attendance. When cases were considered irrespective of anatomic site, the day care OR for Hib disease found in our study was similar to that found for our highest level of crowding in the home (a ratio of two or more people per bedroom relative to less than 1.5 per bedroom) and having three or more children under 6 years of age in the home. Higher family income was an independent marker of risk: There was a protective effect of an annual income greater than $20,000 relative to an income of less than $10,000 that was similar in magnitude to the increased risk associated with day care attendance."

"Although there were no important or statistically significant differences in risk of the different types of Hib disease associated with the other risk factors, the risk associated with day care attendance was almost entirely for Hib meningitis."

"A clear dose-response effect for exposure to day care and Hib meningitis was demonstrated, with increasing risk associated with both increasing hours of day care per week and increasing numbers of children per room. This supports the hypothesis of a direct relation between day care attendance and Hib meningitis."

"In addition to day care attendance, crowding in the home, number of young children in the home, household income, tobacco smoke pollution, and race were independently associated with risk of Hib disease."

"The protective effect of breast feeding in the subgroup of children under 12 months of age was substantial and was statistically significant."

"Although exposure to Hib polysaccharide vaccine appeared to be protective, the effect did not reach statistical significance."

"[A]fter we controlled for number of young children in the home, crowding, maternal education, and income, there was no independent association of tobacco smoke pollution or race with day care."

[32] **"Bed Sharing, Smoking, and Alcohol in the Sudden Infant Death Syndrome," R. Scragg, E.A. Mitchell, B.J. Taylor, A.W. Stewart, R.P.K. Ford, J.M.D. Thompson, E.M. Allen, and D.M.O. Becroft, on behalf of the New Zealand Cot Death Study Group, *British Medical Journal* 307: 1312-1318, 1993**

"We report the first systematic analysis of bed sharing as a risk factor for the sudden infant death syndrome using data collected in a population based case-control study which covered a region with 78% of births in New Zealand. We examined how the risk of sudden infant death from bed sharing is related to other factors, particularly maternal smoking. We tested previous hypotheses -- such as hyperthermia, recent infant illness, and overlaying -- put forward to explain the possible association between bed sharing and sudden infant death. In particular, we examined whether maternal alcohol consumption, implicated in overlaying, is a risk factor by itself or whether it is confounded by other factors such as maternal smoking."

"Our results do not support the idea that infant bed sharing protects against the sudden infant death syndrome. Instead, they show that infant bed sharing is a risk factor for this syndrome, particularly among infants of mothers who smoke. For these infants, the risk of sudden death increased with increasing duration of bed sharing, although it did not vary with the number of cigarettes smoked by the mother."

"In contrast, we found that neither maternal alcohol consumption nor the thermal resistance of the infant's clothing and bedding interacted with bed sharing to increase the risk of sudden infant death; neither was alcohol a risk factor by itself."

"These results have implications as to the likely mechanism(s) by which bed sharing increases the risk of sudden infant death. They do not support a role for overlaying or hyperthermia, for the following reasons. Firstly, if either of these mechanisms was involved the increase in risk of sudden death from bed sharing should have been similar for all infants, regardless of whether the mother smoked. It seems unlikely that maternal smoking would interact with either of these mechanisms to cause an increased risk due to bed sharing only in infants with mothers who smoke."

"Unless there is a third unknown common factor, the interaction between maternal smoking and bed sharing suggests these are components of a sufficient cause that involves a passive smoking mechanisms. Some mothers may be smoking in bed with their infants, although this may not occur commonly because of increasing parental concern in recent years about the effects of passive smoking on children. If few mothers are smoking in bed with their infants, then an alternative explanation for our findings is that rebreathing of expired air from the mother by the infant could lead to hypoxia, as has been postulated to occur in infants dying suddenly who might rebreath [sic] their own expired air when placed prone. . . . Rebreathing may occur, to varying degrees, in all infants who share beds but be most hazardous for infants of smoking mothers."

"Unabsorbed tobacco components from the mother may flow continuously over, and be inhaled by, the baby during sleep, ultimately causing hypoxia and increasing the risk of sudden death. A cumulative exposure by this mechanism would be consistent with the finding of an increased relative risk for infants who usually shared beds in the last two weeks but not in the last sleep."

"If bed sharing is a marker for passive smoking among infants of smoking mothers then our results are consistent with previous epidemiological studies which show that maternal smoking is a risk factor for sudden infant death."

"The observation that paternal smoking did not modify the relative risk from bed sharing was also

unexpected. Perhaps infants are placed on the outside of the bed next to the mother, but away from the father, thus limiting their paternal smoking exposure. . . . Further studies of parental smoking and bed sharing may help to explain why we have observed no effect with fathers and no dose response effect with mothers."

"The attributable risk for cases exposed to both risk factors calculated from our data for bed sharing in the last two weeks suggests that about 20% of all sudden infant deaths in New Zealand can be explained by the joint effect of these two factors. . . . Given the difficulty that many people have in stopping smoking, recommendations that parents who smoke should not share beds with their infants may be more effective in lowering the rate of sudden infant deaths than advising them to stop smoking."

Indoor Air Quality

[33] **"Asthma and the Home Environment," D. Norback, E. Bjornsson, J. Widstrom, G. Strom, C. Edling, U. Palmgren, C. Jansson, and G. Boman. In: *Building Design, Technology, and Occupant Well-Being in Temperate Climates.* E. Sterling, C. Bieva, and C. Collett (eds.). Atlanta, ASHRAE, 329-333, 1993**

"As part of a worldwide investigation of the prevalence of asthma and asthma symptoms, a case-control study was performed in the population of a community in central Sweden. The study comprised 44 subjects with asthma symptoms plus a random sample of 45 subjects without such symptoms. Room temperature, air humidity, volatile organic compounds (VOC), respirable dust, carbon dioxide (CO_2), airborne microorganisms, and mites in settled bedroom dust were measured in the dwellings."

"In a central Swedish urban population, house dust mites were significantly more common in the bedrooms of subjects with asthma symptoms than among controls (OR = 2.5 and 95% CI 1.1-6.1)."

"Furthermore, the average concentration of CO_2 was also higher in the homes of those reporting asthma symptoms, as compared to the controls (960 ppm and 850 ppm, respectively). For other exposure factors, such as air humidity, respirable dust, or relative air humidity, no significant differences could be demonstrated and the average room temperature (68.8_F) was the same in both cases and controls."

"The relation between asthma symptoms and CO_2 concentration was significant even when controlling for the presence of house dust mites....The measurement of volatile organic compounds (VOC) has not yet been evaluated."

"We could also show that normally only about 1% of indoor bacteria and molds in dwellings were viable. The prevalence of dwellings with microbial growth or building moisture was somewhat higher in cases as compared to controls (18% and 13%, respectively), but this difference was not statistically significant."

"In total, 14 of 89 homes (15%) had such signs of microbial growth or building moisture, as judged by an occupational hygienist. No significant differences between asthmatics and controls were found concerning the occurrence of such damp houses."

"House dust mites are frequently found in dwellings in central Sweden, and this can be explained by the fact that indoor air humidity is regularly above 40% even during the winter."

"Our results suggest that the average CO_2 concentration is above the comfort value of 1,000 ppm in many dwellings in Sweden. In addition, we could show that asthmatic symptoms are affected by inadequate outdoor air supply, even when controlling for the presence of house dust mites. Since asthma is a severe disease and is reported to be on the increase in many countries in the western world, three is a need to improve the general ventilation in the dwellings. In order to minimize the occurrence of house dust mites, indoor air humidity should be kept below 40% during the winter and building moisture should be avoided."

[34] **"Experimental Exposures to (1-3)-Beta-D-Glucan," R. Rylander. In: *Building Design, Technology, and Occupant Well-Being in Temperate Climates.* E. Sterling, C. Bieva, and C. Collett (eds.). Atlanta, ASHRAE, 338-340, 1993**

"Previous studies have evaluated the relation between airborne glucan and endotoxins indoors and the extent of subjective symptoms."

"Even if a large number of studies were to show a relation between symptoms and these gents, proof concerning causality can only be obtained in challenge experiments where humans are exposed to the pure substance in concentrations similar to those found in the environment. This report describes the results from preliminary studies on effects after exposure to an aerosol of pure endotoxin and glucan."

"Aerosols of saline, endotoxin, and glucan in saline were produced . . . and introduced into the chamber through the ventilation air ducts about one hour before the subjects entered the chamber. The subjects were exposed during three [four-hour] sessions to saline, endotoxin, or glucan at five- to eight-day interval."

"There were no unexpected reactions or serious symptoms after the different exposures. The most marked reactions were found for nasal and throat irritation. At 1, 4, and 12 hours after exposure, an increase in throat irritation intensity was reported by three to five of the test persons with no difference between the endotoxin and glucan exposures. The intensity increases were statistically significant at 4 hours for nasal irritation...and at 12 hours for throat irritation."

"For symptoms of cough, there was an increase in intensity after endotoxin exposure at 4 and 12 hours but not after glucan exposure. Regarding headache, an increase in symptom intensity was reported only after glucan exposure -- three persons at hours 1 and 4 -- although this increase did not quite reach statistical significance. The intensity of redness of the skin increased at 4 hours after the glucan exposure. There was no increase in eye irritation after endotoxin or glucan exposures."

"In summary, the experience obtained in this first pilot experiment demonstrates that studies of this kind are feasible and that certain of the symptoms reported in sick buildings can be produced by exposure to endotoxin and glucan in amounts that are found in sick buildings. Further experiments are required to increase the size of the exposed group and to study different dose levels of the two compounds. In addition, it is desirable to perform objective tests to assess the exposure effect, such as measurements of pulmonary function or bronchial reactivity."

[35] **"Transformations, Lifetimes, and Sources of NO_2, HONO, and HNO_3 in Indoor Environments,"** C.W. Spicer, D.V. Kenny, G.F. Ward, and I.H. Billick, ***Journal of the Air and Waste Management Association*** 43: 1479-1485, 1993

"Recent research has demonstrated that nitrogen oxides are transformed to nitrogen acids in indoor environments, and that significant concentrations of nitrous acid are present in indoor air. The purpose of the study reported in this paper has been to investigate the sources, chemical transformations and lifetimes of nitrogen oxides and nitrogen acids under the conditions existing in buildings. An unoccupied single family residence was instrumented for monitoring of NO, NO_2, NO_y, HONO, HNO_3, CO, temperature, relative humidity, and air exchange rate. For some experiments, NO_2 and HONO were injected into the house to determine their removal rates and lifetimes. Other experiments investigated the emissions and transformations of nitrogen species from unvented natural gas appliances. We determined that HONO is formed by both direct emissions from combustion processes and reaction of NO_2 with surfaces present indoors. Equilibrium considerations influence the relative contributions of these two sources to the indoor burden of HONO....The lifetimes with respect to reactive processes are on the order of hours for NO and HONO, about an hour for NO_2, and 30 minutes or less for HNO_3. The rapid removal of NO_2 and long lifetime of HONO suggest that HONO may represent a significant fraction of the oxidized nitrogen burden in indoor air."

"There are many possible sources of NO and NO_2 in indoor environments. These species are present in outdoor air, and may infiltrate buildings through normal air exchange processes. Indoor combustion is another source of these species, for example, the use of unvented natural gas, kerosene, or liquified petroleum gas (LPD) appliances, tobacco, and wood burning all contribute to indoor levels of NO and NO_2. Nitrous acid also has been reported in indoor air. Three possible sources of indoor HONO are:

1. Transport of outdoor HONO indoors by air infiltration.
2. Production of HONO by reaction of NO_2 with interior surfaces, with subsequent desorption of the HONO.

3. Direct emission from combustion processes."

"The research reported here demonstrates the importance of chemical reactions occurring in indoor environments for removing nitrogen dioxide from the air and producing gaseous nitrous acid. Our results show that nitrous acid is introduced into indoor air both by heterogeneous NO_2 reactions and by direct emissions from unvented combustion. We suggest that an equilibrium process influences the gas phase nitrous acid concentration, and that surfaces present indoors can serve as a reservoir for HONO."

"The rapid removal of NO_2 due to surface reactions, the formation of HONO by these reactions, and the relatively long lifetime for HONO, suggest that nitrous acid may represent an important fraction of the oxidized nitrogen burden in indoor environments. Because of this, and the recent reports that nitrous acid interferes with many of the methods used to measure NO_2 in indoor environments, it seems likely that some indoor NO_2 data collected in the past may have been influenced by the presence of nitrous acid."

Statistics and Risk Assessment

[36] **"Smoke and Mirrors: The EPA's Flawed Study of Environmental Tobacco Smoke and Lung Cancer," G.L. Huber, R.E. Brockie, and V.K. Mahajan,** ***Cato Review of Business and Government: Regulation*** **1993(3): 44-54, 1993**

"Recently, the Environmental Protection Agency (EPA) completed a report concluding that exposure to environmental tobacco smoke (ETS) -- the residual material from burning cigarettes that is released into indoor air environments by the process of active smoking -- presents a serious and substantial public health problem. The EPA bases its conclusions not on any definitive set of data demonstrating causality, but on a generalized 'total weight of evidence' that, in aggregate, implied causality to the EPA. In reaching those conclusions, the EPA ignored classic criteria for cause-and-effect relationships employed by the scientific community."

"The purpose of this article is to address the more important parts of the EPA report that pertain to adults who are exposed to ETS."

"When the EPA 'speaks,' enormous weight is given to its findings. We generally presume that its conclusions are based on solid scientific evidence and are derived by standard scientific practices."

"Our presumption would be overgenerous in the case of the ETS report, unfortunately. In this case, the EPA's risk assessment is built on the manipulation of data, ignores critical chemical analyses and key epidemiological data, violates time-honored statistical principles, fails to control adequately for important confounding influences (factors other than the one studied that may affect a result or a conclusion) that provide alternative explanations for its conclusions, and violates its own guidelines for assessing and establishing risk to a potential environmental toxin. It lacks credible quality control and adequate external unbiased peer review. In short, in its report on ETS, the EPA did not comply with accepted principles of toxicology, chemistry, and epidemiology, nor with its own guidelines for undertaking cancer risk assessment. In fact, the conclusions drawn by the EPA are not even supported by the EPA's own statements."

"In critically questioning these matters, however, we are not saying that exposure to ETS is without hazard. The data that have been presented in the literature, though, simply do not support any definitive conclusions. We believe that reasonable scientists could interpret the published literature on ETS with differing opinions."

"The EPA states quite authoritatively that 'ETS is a complex mix of over 4,000 compounds.' The EPA states equally clearly that 'this mix contains many known or suspected carcinogens or toxic agents.' Both statements are dubious. No scientific literature supports the assumption that ETS should be treated as a functional equivalent to mainstream smoke."

"What nonsmokers might inhale passively in the presence of smokers is not quantitatively or qualitatively the same material that active smokers inhale from the butt end of a cigarette."

"Is the EPA meta-analysis a scientifically valid manipulation of data? Combining data and undertak-

ing a meta-analysis are valid procedures under appropriate circumstances. But in order to make the outcome value of their meta-analysis 'valid' and 'statistically significant,' the EPA first had to adjust the data as originally published in peer-reviewed literature and, second, they had to broaden the confidence intervals to a scientifically unconventional level of 90 percent."

"When a number of studies are combined, the confidence intervals generally are 'ratcheted down,' or tightened, to assess significance; the EPA did just the opposite and in so doing diminished its report's scientific value. Lowering of statistical standards to make valid otherwise unmeaningful results is an unusual and dubious scientific practice....The manipulation of data in this manner to develop statistical significance permitted the EPA to declare passive smoking a Group A carcinogen -- the highest rank possible. Without the recalculations and manipulations, the EPA would have not met any of the three classic criteria for establishing risk."

"The EPA's risk assessment acknowledged that confounders are important to any evaluation of ETS as a potential carcinogen. Its concern for confounders was extremely limited, however, and their influence was evaluated by employing a modeling of data by a method as yet untested and unproved by conventional peer review. The EPA, in essence, ignored its own guidelines and established requirements to rule out confounding as an alternative explanation for an association before basing causal inference on epidemiologic results."

"Scientific integrity was compromised, if not outright abused, by the manner in which this risk assessment was generated....Science should dictate what policies need to be established; predetermined policies should not dictate how science should be interpreted....Will reality and fact ever catch up with political science at the EPA?"

APPENDIX B

UPCOMING SCIENTIFIC MEETINGS

- **February 14, 1994**
  One-Day IAQ Course, Environmental Law and Policy Program, George Washington University, Washington, D.C. [In This Issue] Same program to be held September 7, 1994
- **March 4, 1994**
  Indoor Air Quality: An Overview for People Who Need to Know, AIHHM, San Antonio, Texas [Issue 57, Item 35] Same program to be held April 13, 1994, Minneapolis, Minnesota; May 5, 1994, Chicago, Illinois; June 17, 1994, Oklahoma City, Oklahoma; July 14, 1994, Anchorage, Alaska
- **March 22-24, 1994**
  Indoor Environment '94, IAQ Publications and other sponsors, Washington, D.C. [Issue 61, Item 30]
- **March 28-31, 1994**
  Eleventh ORNL Life Sciences Symposium, Indoor Air and Human Health Revisited (Bringing Selected Advances in Medical Science to the Indoor Air Quality Community), Knoxville, Tennessee [Issue 58, Item 43]
- **May 5-7, 1994**
  Second Annual IAQ Conference and Exposition, NCIAQ, Tampa, Florida [Issue 49, Item 35]
- **May 22, 1994**
  Indoor Air Quality Symposium, American Industrial Hygiene Conference and Exposition, Anaheim, California [Issue 57, Item 34]
- **August 22-25, 1994**
  Healthy Buildings '94, Budapest, Hungary [In This Issue]
- **October 10-14, 1994**
  9th World Conference on Tobacco and Health, Paris, France [Issue 60, Item 38]
- **October 18-20, 1994**
  Indoor Air Quality in Asia, Beijing, China [Issue 54, Item 42]
- **October 30-November 2, 1994**
  IAQ '94: Engineering Indoor Environments, ASHRAE and other sponsors, St. Louis, Missouri [Issue 58, Item 42]